Luke's Hot Pots → Mountain Spaa

Luke's Hot Spa

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Annotations

Treatment of bradycardia and hypotension syndrome in patients with acute myocardial infarction

The practical clinical management of arrhythmias and cardiovascular failure in patients with acute myocardial infarction is becoming increasingly important with the advent of intensive care and coronary units. Although the occurrence of bradycardia and associated cardiovascular failure has long been recognized by clinicians, analysis of the hemodynamic changes underlying clinical syndromes and evaluation of treatment has only recently received detailed analysis.

Sinus bradycardia occurs at some time in approximately 20 per cent of patients with acute myocardial infarction. It usually becomes apparent within the first hours of illness and may be spontaneous, the result of morphine[1] or methadone administration, or may be related to extreme pain. The spontaneous form is seen particularly in patients with posterior infarctions[2] but may also occur when the infarct is anterior. Ventricular bradycardia is often associated with a short P-R interval, abnormal P waves, A-V dissociation, nodal rhythm, and second-degree A-V block.

When bradycardia is moderate (50 to 60 per minute) and transient, it is usually of little clinical significance. More serious consequences follow if bradycardia is profound (25 to 40 per minute) and particularly if ventricular function is poor with low stroke volume. Persistently low heart rate leads to fall in cardiac output and arterial pressure. The associated clinical syndromes vary in severity. Symptoms may be limited to a weak sensation and nausea while lying flat, but with dizziness, pallor, and sweating after sitting up or standing. The symptoms and general appearance of the patient resemble those of a common "vasovagal" faint. If the changes progress, mental confusion may be succeeded by loss of consciousness associated with the clinical picture of profound circulatory "shock." Cardiac arrest may follow.

Treatment is indicated in most patients at an early stage in view of the potential hemodynamic deterioration and also because of the increased incidence of serious arrhythmias occurring in patients in whom bradycardia is allowed to persist.[3] Mild cases may be managed conservatively by maintaining the patient flat in bed or by raising the foot of the bed. If the heart rate does not increase and blood pressure remains low, atropine sulfate injected intravenously is often effective.[4] In the first place, 0.3 mg. should be given followed by further 0.3 mg. increments up to a total of 2 mg.: "titrating" the patient to a heart rate of 80 to 90 per minute. Subsequent doses are required every 3 to 4 hours. It must be emphasized that the drug should be given intravenously as absorption from subcutaneous or intramuscular injection is too slow and unpredictable under conditions of circulatory failure.

In the case of rapid clinical deterioration, the patient should lie flat and the legs should be raised 45 degrees to the horizontal by an assistant simultaneous with the administration of atropine. Under these circumstances, isoproterenol, 0.05 mg. intravenously, is also effective and is often superior to atropine in the presence of second-degree heart block. With less urgent situations, isoproterenol should be given by intravenous infusion (1 mg. per 500 ml. of dextrose) for better control and avoidance of overdose.[3] Drug therapy of the bradycardia hypotension syndrome may often be withdrawn after approximately 12 hours, but any tendency to recur should be treated at an early stage.

The physiological mechanisms involved in bradycardia and associated cardiovascular failure are not clearly understood. Slowing of the heart may be due to a direct effect of the infarct on the pacemaker tissues. Alternatively, there may be a disturbance of parasympathetic and sympathetic autonomic activity. This is supported by preliminary measurements of urine catecholamines which show low levels of excreted norepinephrine when the heart rate is low.[5] Reflex effects could be mediated directly from receptors in the heart,[6,7] or could involve more complex nervous activity secondary to pain and anxiety. Bradycardia hypotension syndromes following morphine administration are also probably the result of central nervous system reflex activity as suggested by analytical experiment.[8]

The mechanisms by which atropine and isoproterenol benefit the circulation are probably concerned to a large extent with increasing cardiac output and arterial pressure. Atropine[4] increases cardiac output in proportion to heart rate, the stroke volume remaining constant. Some increase in stroke volume may occur when the patients' legs are raised at the same time. Arterial pressure rise is less marked than cardiac output owing to a fall in peripheral resistance. Isoproterenol improves cardiac output both by an increase in heart rate and probably of stroke vol-